

In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: February 26, 2021

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RUSELL BLENDER,

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PUBLISHED

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Petitioner,

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No. 16-1308V

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v.

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Special Master Nora Beth Dorsey

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SECRETARY OF HEALTH
AND HUMAN SERVICES,

*

Entitlement; Influenza (“Flu”) Vaccine;

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Pneumococcal Conjugate (“Pprevnar 13” or

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“Pprevnar”) Vaccine; Polyneuropathy;

Respondent.

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Chronic Idiopathic Axonal Polyneuropathy

*

(“CIAP”).

* * * * *

Amber D. Wilson, Wilson Science Law, Washington, DC, for petitioner.

Althea W. Davis, U.S. Department of Justice, Washington, DC, for respondent.

RULING ON ENTITLEMENT¹

I. INTRODUCTION

On October 11, 2016, Rusell Blender (“petitioner”) filed a petition under the National Vaccine Injury Compensation Program (“Vaccine Act” or “the Program”), 42 U.S.C. § 300aa-10 et seq. (2012).² Petitioner alleged he suffered a polyneuropathy as a result of an influenza (“flu”) vaccine administered on November 4, 2013 and a pneumococcal conjugate (“Pprevnar 13” or “Pprevnar”) vaccine administered on November 14, 2013. Petition at 1-2 (ECF No. 1). Respondent argued against compensation, stating that the case was “not appropriate for

¹ Because this Ruling contains a reasoned explanation for the action in this case, the undersigned is required to post it on the United States Court of Federal Claims’ website in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services). **This means the Ruling will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access.

² The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2012). All citations in this Ruling to individual sections of the Vaccine Act are to 42 U.S.C. § 300aa.

compensation under the terms of the Act.” Respondent’s Report (“Resp. Rept.”) at 1 (ECF No. 25).

After carefully analyzing and weighing the evidence in accordance with the applicable legal standards, the undersigned finds that petitioner provided preponderant evidence that the flu vaccine petitioner received caused him to develop a polyneuropathy, which satisfies his burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, petitioner is entitled to compensation.

II. ISSUES IN AGREEMENT AND IN DISPUTE

The parties agree that petitioner’s polyneuropathy is best characterized as chronic idiopathic axonal polyneuropathy (“CIAP”). Pet. Motion for Findings of Fact and Conclusions of Law (“Pet. Mot.”), filed June 1, 2020, at 5, 8 (ECF No. 81); Resp. Response to Pet. Mot. (“Resp. Response”), filed Oct. 26, 2020, at 20 (ECF No. 93). However, they disagree as to whether the November 2013 flu and Prevnar vaccinations caused his condition. Pet. Mot. 5-6, 8, 24; Resp. Response at 20-25.

III. CHRONIC IDIOPATHIC AXONAL POLYNEUROPATHY

Peripheral neuropathy is a “disorder of the peripheral nervous system, which may be associated with varying combinations of weakness, autonomic changes, and sensory changes.” Resp. Exhibit (“Ex.”) D2 at 1.³ “The term peripheral neuropathy refers to any disorder of the peripheral nervous system including single and multiple (asymmetric) mononeuropathy, and symmetrical involvement of many nerves (polyneuropathy).” Pet. Ex. 22 at 1.⁴ Most peripheral neuropathies are chronic in nature and develop over a period of several months. Id. Neuropathies are “broadly classified into small or large fiber neuropathies,” with unmyelinated and thinly myelinated fibers referred to as small fibers and myelinated fibers called large fibers. Id.

CIAP is a term used to describe “neuropathies with both sensory and motor involvement in a length depend[en]t distribution where neurophysiology reveals axonal damage, neuropathy onset is insidious and shows slow or no progression of the disease over at least 6 months with no [etiology being identified despite appropriate investigations.” Pet. Ex. 22 at 1. The term CIAP

³ Ling Han et al., Peripheral Neuropathy Is Associated with Insulin Resistance Independent of Metabolic Syndrome, 7 Diabetology & Metabolic Syndrome 1 (2015).

⁴ Panagiotis Zis et al., Chronic Idiopathic Axonal Polyneuropathy: A Systematic Review, 263 J. Neurology 1903 (2016). Polyneuropathy, also called a peripheral neuropathy, is a “neuropathy of several peripheral nerves simultaneously.” Polyneuropathy, Dorland’s Online Med. Dictionary, <https://www.dorlandsonline.com/dorland/definition?id=40203> (last visited Feb. 17, 2021).

has been adopted by some researchers, while others use cryptogenic⁵ sensory peripheral neuropathy (“CSPN”) and other like terms to describe such patients.⁶ Pet. Ex. 19 at 5, 5 n.1; see Pet. Ex. 23.⁷

Patients with CIAP exhibit slow progression and have mild initial symptoms. Pet. Ex. 21 at 1-2;⁸ Pet. Ex. 22 at 4. Patients with CIAP can exhibit both sensory and motor symptoms. Pet. Ex. 22 at 3. “Sensory symptoms include[] tingling, pins and needles, numbness, tightness, burning, pain[,] and sensory ataxia.” Id. “Motor symptoms include[] muscle cramps, stiffness, weakness[,] and wasting.” Id. Sensory symptoms are more prominent in CIAP patents, with the most commonly reported symptom being numbness. Id. Sensory symptoms “occur in a roughly symmetrical pattern in the distal lower extremities or upper extremities or both and evolve over weeks to months.” Pet. Ex. 23 at 3.

IV. BACKGROUND

A. Procedural History

Petitioner filed his petition requesting compensation under the Vaccine Act on October 11, 2016. Petition at 1. The undersigned held an initial status conference on November 29, 2016, where the parties discussed petitioner’s medical records and next steps. Order dated Nov. 29, 2016 (ECF No. 7). Between November 2016 and May 2017, petitioner filed medical records. Pet. Exs. 1-12. On August 25, 2017, respondent filed his Rule 4(c) Report, recommending against compensation. Resp. Rept. at 1. Petitioner filed additional medical records and an affidavit on October 13, 2017. Pet. Exs. 13-18.

On January 19, 2018, petitioner filed an expert report of Dr. Raji Grewal. Pet. Ex. 19. Respondent filed an expert report of Dr. Vinay Chaudhry on May 21, 2018. Resp. Ex. A.

The undersigned held a Rule 5 conference on August 13, 2018, where she provided the parties with her preliminary opinions. Rule 5 Order dated Aug. 13, 2018 (ECF No. 40). The undersigned determined that based on the expert reports and her review of the medical records, petitioner likely suffered from a polyneuropathy. Id. at 1. The undersigned further found petitioner could likely meet his burden under Althen Prongs One and Two, but she required additional information on onset before making a preliminary determination regarding Althen

⁵ Cryptogenic means idiopathic. Cryptogenic, Dorland’s Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=11845> (last visited Jan. 28, 2021).

⁶ While CIAP may also be referred to as CSPN, the undersigned will use the phrase CIAP throughout this Ruling.

⁷ Mamatha Pasnoor et al., Cryptogenic Sensory Polyneuropathy, 31 Neurologic Clinics 463 (2013).

⁸ R. A. C. Hughes et al., A Controlled Investigation of the Cause of Chronic Idiopathic Axonal Polyneuropathy, 127 Brain 1723 (2004).

Prong Three. Id. at 1-2. Petitioner filed additional documentation on August 27 and October 5, 2018. Pet. Exs. 24-27.

On September 20, 2018, the undersigned held a status conference at the request of respondent's counsel to discuss respondent's concerns with the undersigned's Rule 5 Order. Order dated Sept. 24, 2018, at 1 (ECF No. 42). Considering the experts' wide variety of terms used to describe petitioner's condition, the undersigned ordered the parties to file supplemental expert reports explaining how they would define petitioner's neuropathy and whether petitioner's condition was immune-mediated. Id. Respondent filed his supplemental expert report by Dr. Chaudhry on November 20, 2018, and petitioner filed an expert report by Dr. Enrique Aradillas on April 18, 2019. Resp. Ex. C; Pet. Ex. 28.

On July 19, 2019, respondent filed a status report, indicating that he was not interested in pursuing settlement negotiations, and a supplemental expert report from Dr. Chaudhry. Resp. Status Rept., filed July 19, 2019 (ECF No. 61); Resp. Ex. D. Petitioner filed a supplemental expert report by Dr. Aradillas as well as a Motion to Close the Evidentiary Record on November 25, 2019. Pet. Ex. 50; Pet. Mot. to Close the Evidentiary Record and to Set Deadlines for Case Resolution ("Mot. to Close Record"), filed Nov. 25, 2019 (ECF No. 69). Respondent filed his response on January 8, 2020, and petitioner filed a reply on January 15, 2020. Resp. Response to Mot. to Close Record, filed Jan. 8, 2020 (ECF No. 73); Pet. Reply to Resp. Response to Mot. to Close Record, filed Jan. 15, 2020 (ECF No. 74).

The undersigned held a status conference on January 28, 2020 to discuss the next steps in this matter. Order dated Jan. 28, 2020 (ECF No. 75). The undersigned opined that a hearing was unnecessary, and set a briefing schedule to resolve this case on the record. Id. at 1. On June 1, 2020, petitioner filed his Motion for a Ruling on the Record. Pet. Mot. Respondent filed his response to petitioner's Motion on October 26, 2020, and petitioner filed his reply on December 21, 2020. Resp. Response; Pet. Reply to Resp. Response ("Pet. Reply"), filed Dec. 21, 2020 (ECF No. 96).

This matter is now ripe for adjudication.

B. Summary of Relevant Facts

At seventy years old, petitioner received a flu vaccine on November 4, 2013 and a Prevnar vaccine on November 14, 2013. Pet. Ex. 1 at 1. Petitioner's past medical history was significant for Gilbert syndrome,⁹ benign prostatic hypertrophy, hypogonadism, osteoporosis, degenerative arthritis of his left hip, and a closed fracture of the wrist. Resp. Rept. at 2; Pet. Ex. 2 at 5, 47; Pet. Ex. 7 at 2.

⁹ Gilbert syndrome is a genetic "disorder of bilirubin metabolism." Gilbert Syndrome, Dorland's Med. Dictionary Online, <https://www.dorlandonline.com/dorland/definition?id=110655> (last visited Feb. 8, 2021).

On December 3, 2013, petitioner saw his primary care physician (“PCP”), Dr. Scott Adler, for a follow-up visit.¹⁰ Pet. Ex. 2 at 2. He presented with a sore throat for the past two weeks that began after a dentist appointment.¹¹ Id. He was assessed with a sore throat and prescribed Sucralfate, an antacid. Id. at 3-4. There is no indication that petitioner complained of neurological symptoms at this visit. See id. at 2-4.

On December 10, 2013, petitioner had a follow-up visit to his endocrinologist, Dr. Ned M. Weiss. Pet. Ex. 3 at 8. Petitioner was noted to have “[s]ome rash – definitely related to androgel.” Id.

Petitioner presented to Dr. Samir Undavia for an evaluation of his throat on December 26, 2013. Pet. Ex. 11 at 1. Petitioner complained of a sore throat affecting his eustachian tube and heartburn. Id. On examination, Dr. Undavia noted a normal neurologic exam. Id. Petitioner was assessed with septal deviation, turbinate hypertrophy, allergic rhinitis, and cough secondary to reflux esophagitis, and prescribed a Medrol dose pack and omeprazole.¹² Id. at 3.

According to petitioner’s pharmacy records, petitioner purchased the Medrol pack and omeprazole on December 26, 2013. Pet. Ex. 24; Pet. Ex. 27 at ¶ 2. Petitioner averred that he “began taking the prescribed medication on December 27, 2013.” Pet. Ex. 27 at ¶ 3.

Petitioner returned to Dr. Undavia for a follow-up exam on January 16, 2014. Pet. Ex. 11 at 3. Petitioner reported that he completed the Medrol dose pack and “after the second night of taking the [o]meprazole [he] started getting a numbness and tingling on the bottom of his toe joints,” so he stopped taking it. Id. He further “report[ed] that his throat issue [] resolved” and that he felt “90% better with medical management.” Id.

Petitioner called his PCP, Dr. Adler, on January 17, 2014 to provide him with an update. Pet. Ex. 13 at 1. Petitioner reiterated that he felt a tingling sensation after the second day of taking omeprazole. Id. Petitioner called Dr. Adler again on January 22, 2014 to note the numbness of his feet was better. Id. However, on January 30, 2014, petitioner called Dr. Adler complaining of continued paresthesia. Id. at 2.

On May 20, 2014, petitioner’s PCP noted that petitioner was still experiencing tingling and an electromyogram (“EMG”) was recommended. Pet. Ex. 13 at 2.

¹⁰ It is not clear from the records when petitioner’s original visit to Dr. Adler took place.

¹¹ According to petitioner’s dental records, he had a dentist appointment on November 20, 2013. Pet. Ex. 15 at 2.

¹² Omeprazole is a proton pump inhibitor (“PPI”) used in the treatment of gastroesophageal reflux disease. Omeprazole, Dorland’s Med. Dictionary Online, <https://www.dorlands.com/dorland/definition?id=34985> (last visited Feb. 8, 2021). A PPI is “an agent that inhibits the proton pump in the stomach, thus limiting gastric acid secretion.” Proton Pump Inhibitor, Dorland’s Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=82780> (last visited Feb. 8, 2021).

On June 5, 2014, petitioner presented to neurologist, Dr. Chitharanjan Rao, upon referral from Dr. Adler. Pet. Ex. 17 at 6. Dr. Rao's record documented that petitioner complained of numbness and tingling in his feet since January 2014, which began "a few days after" he was prescribed medication for GERD. Id. He reported a "slight worsening" of symptoms. Id. Petitioner had no sensory symptoms in his hands and no loss of balance when his eyes were closed, but he did think he was "slightly more unsteady when he [was] standing on one leg." Id. Petitioner's neurological symptom review was otherwise unremarkable. Id. Dr. Rao found petitioner's neurological exam consistent with "[m]ild, distal, symmetric sensory (small and large fiber) polyneuropathy" and cervical myelopathy. Id. at 7. Dr. Rao ordered an EMG, MRI of petitioner's cervical spine, and laboratory tests to determine the etiology of his peripheral neuropathy. Id.

Laboratory studies were performed on June 11, 2014, and showed the following results: Immunoglobulin G ("IgG") was 877 (within normal range); Immunoglobulin A ("IgA") was 75 (low); and Immunoglobulin M ("IgM") was 64 (normal).¹³ Pet. Ex. 8 at 13. Glucose tolerance tests were normal. Id. at 14. Hepatitis and Lyme disease testing were negative, Vitamin B12 and Folate results were normal, and Anti-Hu Antibodies¹⁴ were negative. Id. at 14-15. Hemoglobin A1c was normal, indicating that petitioner was not at risk for diabetes. Id. at 15. Rheumatoid Arthritis Factor was normal, and Antinuclear Antibodies Direct was negative. Id. Serum and urine immunofixation lab studies were done on June 13, 2014, and interpreted as normal. Id. at 11-12.

Dr. Rao next saw petitioner on June 18, 2014. Pet. Ex. 8 at 8. Electrodiagnostic exams, including an EMG, were performed. Id. at 8-10. The EMG, interpreted by Dr. Rao, showed evidence of a "[m]ild, large fiber, length-dependent, sensory axonal polyneuropathy" and a "[m]ild, right ulnar mononeuropathy at the elbow, with no axon loss to the innervated muscles."¹⁵ Id. at 10. "No electrodiagnostic evidence of primary muscle disease, bilateral lumbosacral radiculopathy[,], or right cervical radiculopathy" was noted. Id.

¹³ Immunoglobulins are "structurally related glycoproteins that function as antibodies." Immunoglobulin, Dorland's Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=24894> (last visited Feb. 10, 2021). IgA antibodies protect "against infection in mucosal areas of the body such as the respiratory tract (sinus and lungs) and the gastrointestinal tract (stomach and intestines)." Immunoglobulins (IgA, IgG, IgM), Merck Manual, <https://www.merckmanuals.com/-/media/Manual/LabTests/ImmunoglobulinsIgAIgGIgM> (last visited Feb. 10, 2021). IgM antibodies "are produced as a body's first response to a new infection," and "increase for several weeks and then decline as IgG production begins." Id. IgG antibodies "are produced during an initial infection or other antigen exposure, rising a few weeks after it begins, then decreasing and stabilizing." Id.

¹⁴ "Antibodies to Hu antigen also called antineuronal nuclear antibody (ANNA1) are present in patients with paraneoplastic neurologic syndrome such as Encephalomyelitis and are frequently associated with small-cell lung cancer" and other forms of cancer. Pet. Ex. 8 at 15.

¹⁵ The EMG finding of right ulnar mononeuropathy at the elbow is not at issue in petitioner's vaccine injury claim.

On June 20, 2014, petitioner saw Dr. Rao for a follow-up exam. Pet. Ex. 8 at 18. Dr. Rao documented that, “[petitioner] began experiencing tingling and numbness of his toes involving the ventral surfaces bilaterally since January 2014. He was prescribed [medication] for GERD in January 2014, [and] he thinks his symptoms began a few days after.” Id. Dr. Rao concluded that petitioner’s neurological exam was consistent with “[m]ild, distal, symmetric sensory (small and large fiber) polyneuropathy” and cervical myelopathy.¹⁶ Id. at 19. The laboratory and bloodwork previously performed did not reveal the etiology of petitioner’s condition. Id. Although Dr. Rao did not determine the cause of the petitioner’s polyneuropathy, he opined that the three potential (or differential) causes were, “metabolic, toxic and immunological.” Id.

Petitioner underwent an initial physical therapy (“PT”) evaluation on July 25, 2014. Pet. Ex. 10 at 7. Petitioner reported an insidious onset of a left, lower extremity neuropathy which began January 2014. Id. Petitioner reported that taking a Medrol Dosepak correlated to the onset of his neuropathy. Id. After ten PT sessions, petitioner was discharged on August 15, 2014. Id. at 4. His progress upon discharge was good, and he was advised to continue at-home exercises. Id.

On November 17, 2014, petitioner saw his PCP for his annual health assessment. Pet. Ex. 13 at 12. Dr. Adler noted petitioner’s “[t]ingling feeling in legs has gradually progressed generally up to the lower calves though occasionally higher,” and his “left leg strength has improved but [he] is somewhat more tired at the end of the day.” Id. Petitioner was noted to have no neurologic symptoms in his upper extremities. Id. On exam, Dr. Adler found “pinprick slightly decreased [in] left first toe” and “tandem gait intact.” Id. at 15. He explained that petitioner’s neuropathy “has been extensively evaluated” and “[t]here was only a minimal abnormality on the porphyria test which [he] discussed with hematology who felt it was unlikely to be significant.” Id. at 16.

Petitioner presented to Dr. Adler, on May 19, 2015, for a follow-up visit. Pet. Ex. 2 at 5. Under his review of symptoms, petitioner was “[p]ositive for paresthesia (bilateral lower extremity; feels it is progressing up to above ankle).” Id. Petitioner’s neurological exam showed “normal [deep tendon reflexes] elicited in biceps, triceps, supinator, knee, and ankle jerk.” Id. at 7. Sensory examination was noted to be “normal to light touch, monofilament, and pinprick,” and “no focal weakness [was] noted.” Id. Petitioner’s coordination was normal, his Romberg test was negative, and he had a tandem gait. Id. Dr. Adler’s assessment was “[o]ther specified idiopathic peripheral neuropathy.” Id.

Petitioner called his PCP on July 13, 2015, complaining of some tingling in his left calf and a vibrating feeling in both legs the prior night. Pet. Ex. 13 at 3. He also felt his heart rate was fast and urine was a darker yellow. Id.

On May 17, 2016, petitioner saw his PCP for a follow-up visit. Pet. Ex. 2 at 9. Petitioner “present[ed] with history of other specified idiopathic peripheral neuropathy. [F]eels tingling on

¹⁶ Dr. Rao opined that petitioner’s cervical myelopathy was more likely due to a “compressive (spondylotic)” cause. Pet. Ex. 8 at 18.

most of bottom of feet, [occasionally] higher.” Id. On exam, his sensory examination was “normal to light touch, monofilament, and pinprick” and “no focal weakness noted.” Id. at 11.

Petitioner next saw Dr. Adler on November 30, 2016, for his annual health assessment. Pet. Ex. 13 at 4. Dr. Adler noted petitioner was “generally feeling well” and his neuropathy had plateaued. Id. Petitioner’s “[p]aresthesias in legs seems to have stabilized around the level of upper calves.” Id. Petitioner also “feels left leg is slightly stronger than a year ago.” Id. Under past medical history, Dr. Alder noted, “[n]europathy, possibl[y] related to flu shot.” Id. at 5.

On December 14, 2016, petitioner saw rheumatologist, Dr. Richard Gordon. Pet. Ex. 9 at 20. Dr. Gordon noted that petitioner reported that “[h]e developed a rash or perhaps hives after a dental cleaning in November” and “then developed discomfort and swelling of his right volar wrist.” Id. The discomfort became generalized in both hands and petitioner currently has a problem with his right ankle and discomfort of the right inner thigh. Id. Petitioner denied tick exposure, infectious disease exposure, foreign travel, unusual fever, or other systemic complaints. Id. On exam, Dr. Gordon noted “there is nothing to be seen. There might be slight tenderness of the right second digit but not specifically the PIP or MCP although the MCPs of both hands were somewhat tender with compression.” Id. He also noted petitioner’s wrists and knees were unremarkable, his right hip had good mobility, and his left hip had limited internal rotation. Id. Dr. Gordon found petitioner’s history of peripheral neuropathy “unexplained,” and found it possibly related to his Medrol/Prilosec¹⁷ medications.¹⁸ Id. He indicated that he was “not really sure what is going on,” but finds petitioner “almost has a polyarthropathy.” Id. at 21. Further labs and X-rays were ordered, and petitioner was prescribed Aleve. Id.

On December 15, 2016, petitioner underwent an X-ray on his right hand. Pet. Ex. 9 at 5. The impression was “[m]ild degenerative changes, most notably right fifth DIP joint.” Id. Petitioner presented to Dr. Gordon for a follow up evaluation on December 21, 2016. Id. at 22. Petitioner reported taking Aleve and Tylenol with improvement in his sleep quality. Id. Petitioner also had “discomfort in both hands as well as the right ankle among other areas.” Id. Dr. Gordon wrote, petitioner’s “symptoms remain about the same or perhaps slightly less.” Id. Laboratory test were unremarkable. Id. Dr. Gordon diagnosed petitioner with “a seronegative arthropathy,” with early rheumatoid arthritis as a possibility. Id. Because Dr. Gordon was nervous about prescribing medications, he recommended a “watch and wait” approach. Id.

Dr. Adler saw the petitioner on June 5, 2017, for a follow up of his idiopathic peripheral neuropathy. Pet. Ex. 13 at 60. Under review of symptoms, there is no documentation that petitioner reported numbness or tingling. Id. Physical exam was normal. Id.

¹⁷ Prilosec is a “trademark for a preparation of omeprazole.” Prilosec, Dorland’s Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=40925> (last visited Feb. 8, 2021).

¹⁸ Although Dr. Gordon suggested the possibility of an association between petitioner’s medications (Medrol and omeprazole) and his polyneuropathy, that association was not made by petitioner’s neurologists, or either parties’ experts. No physician expressed an opinion that petitioner’s polyneuropathy was caused by either of these medications.

Petitioner next saw Dr. Adler on December 7, 2017. Pet. Ex. 26 at 8. Dr. Adler noted petitioner was doing well other than his neuromuscular issues. Id. Under review of symptoms, Dr. Adler wrote that petitioner's paresthesias had progressed. Id. Petitioner "sporadically has some dysesthesia in the left heel and tip of right toe" and has "more pronounced paresthesias around the rim of left foot." Id. "Neuropathy, possibility related to flu shot" remained in the description of petitioner's past medical history. Id. at 9. Dr. Adler found "no dramatic change" in petitioner's neuropathy, but noted it appeared to "symptomatically progress a bit." Id. at 12. He noted no further testing was indicated and recommended that petitioner consider seeing a neurologist again for additional treatments. Id.

On May 1, 2018, petitioner returned to Dr. Adler complaining of pain and discomfort in forearm and upper left back for a few days and "pins and needles" for ten minutes in his left calf when moving from chair to bed. Pet. Ex. 26 at 3. Petitioner reported his right toe sometimes hurts. Id. at 4. Petitioner also reported tingling, but no burning, weakness, or numbness. Id.

C. Expert Reports

1. Petitioner – Dr. Raji Grewal¹⁹

a. Background and Qualifications

Dr. Grewal is a clinical neurologist and board certified in neurology and neuromuscular medicine. Pet. Ex. 19 at 1. After obtaining his M.D. from the University of Alberta in 1982, he completed a rotating internship and residency in internal medicine in Canada, as well as a residency in neurology at UCLA School of Medicine and a fellowship in the Department of Neurology at Columbia University. Pet. Ex. 20 at 1-2. Dr. Grewal is a Professor of Neuroscience at Seton Hall University and a clinical neurologist at the Neuroscience Institute at St. Francis Medical Center in New Jersey. Pet. Ex. 19 at 1; Pet. Ex. 20 at 4. His practice focuses on the medicine of neuromuscular disorders. Pet. Ex. 19 at 1. He has served on various committees and authored or co-authored almost 150 publications. Pet. Ex. 20 at 9, 11-28.

b. Opinion

Dr. Grewal concluded that to a reasonable degree of medical certainty, the flu and Prevna vaccinations administered to petitioner were a "substantial cause or trigger of the onset of neuropathy," leading to his residual numbness. Pet. Ex. 19 at 1.

Based on petitioner's medical history, neurological examination, and EMG, Dr. Grewal opined that petitioner developed an idiopathic peripheral neuropathy. Pet. Ex. 19 at 3. He defined peripheral neuropathy as "the range of clinical syndromes affecting a variety of peripheral nerve cells and fibers, including motor, sensory, and autonomic fibers." Id. at 4. When a neuropathy cannot be linked to a known or identifiable cause, it is often labeled as idiopathic. Id. However, he noted that "idiopathic" does not mean "no identifiable biologically plausible cause," but simply means "there is a biological cause that just has not been identified."

¹⁹ Petitioner filed one expert report authored by Dr. Grewal. Pet. Ex. 19.

Id. at 4, 6. Dr. Grewal, citing Hughes et al., pointed out that no identifiable cause is found in 10-40% of patients with peripheral neuropathies. Id. at 4 (citing Pet. Ex. 21 at 1).

Dr. Grewal explained that the most common acute peripheral neuropathies are demyelinating or axonal. Pet. Ex. 19 at 4. Demyelinating neuropathies, like Guillain-Barré Syndrome (“GBS”), are those where the myelin sheath around the nerves is destroyed. Id. Axonal neuropathies, on the other hand, are characterized by a “loss of motor or sensory nerve axons.” Id. Both have “overlapping features, including an autoimmune etiology.” Id. at 4-5.

Dr. Grewal opined that based on petitioner’s clinical course and EMG, petitioner suffered a loss of motor or sensory nerve axons, indicating that he developed an acute axonal sensory peripheral neuropathy. Pet. Ex. 19 at 4-5. A subset of patients are described as having a “slowly progressive axonal sensory and/or motor neuropathy,” termed CIAP. Id. at 5. CIAP “refers to patients who have a symmetrical, length dependent peripheral neuropathy primarily characterized by axonal damage involving large fibers, insidious onset, slow progression, [where] no other etiology can be identified.” Id.

Petitioner’s neuropathy, Dr. Grewal opined, is immune-mediated and “initiated by cross-reactivity of certain immune cells, from an initial immunologic response to an immune challenge (e.g., a virus, a bacterium, or a vaccine), and transmuting to inadvertent immune response against self cells. Structural similarities between microbial and self-peptides can result in the activation of autoreactive T cells,” a process referred to as molecular mimicry. Pet. Ex. 19 at 5 (internal quotation marks omitted). For support, Dr. Grewal cited Zis et al., a systematic review of CIAP, that noted reports of T cells and antibodies in patients with CIAP, to support his opinion that the condition can have an autoimmune etiology. Id. at 6 (citing Pet. Ex. 22 at 6).

Dr. Grewal explained that although the acute onset of petitioner’s neuropathy is unusual, no cause was identified in petitioner’s testing and no temporally related infection was found. Pet. Ex. 19 at 4, 6. He concluded that petitioner’s vaccinations were more likely than not substantial factors in causing his symptoms because (1) no other trigger of petitioner’s symptoms was found and (2) immune-mediated nerve damage is a known cause of neuropathies. Id. at 6.

Dr. Grewal opined that the temporal association between petitioner’s vaccinations and the onset of his neurological symptoms was medically appropriate. Pet. Ex. 19 at 6. He explained that a medically appropriate timeframe for onset of clinical symptoms after an immune challenge ranges from two to 60 days. Id. Petitioner reported an onset at the end of December 2013, which is within this timeframe.²⁰ Id. at 7.

Dr. Grewal concluded that the vaccinations most likely led to petitioner producing antibodies, which in turn attacked his axonal neurons. Pet. Ex. 19 at 7. He found “[i]t is most likely that [petitioner] responded to his vaccinations by producing antibodies that then attacked his axonal nerves,” and thus, “[i]t is certainly plausible that axonal damage was occurring from the production of autoantibodies for several weeks before [petitioner] began to feel numbness

²⁰ Dr. Grewal deferred to an immunologist to describe the implication of having two vaccinations within a medically appropriate timeframe. Pet. Ex. 19 at 7.

and tingling.” Id. Therefore, he found “it is more likely than not the vaccinations [] caused a process of immune-mediated, inflammatory, sensory polyneuropathy.” Id.

2. Petitioner – Dr. Enrique Aradillas²¹

a. Background and Qualifications

Dr. Aradillas is a board certified neurologist. Pet. Ex. 28 at 1. He obtained his M.D. in 2003 in Mexico, and thereafter completed an internal medicine residency, neurology residency, and a fellowship in interventional pain management. Pet. Ex. 29 at 1. Dr. Aradillas currently works as the director of the Neuropathic Pain Center at the Vincera Institute, where he evaluates and treats patients with chronic neuropathic pain, painful peripheral neuropathies, and underlying common chronic painful conditions. Pet. Ex. 28 at 1. He is also in charge of ongoing research collaborations with Drexel University. Id. He has authored or co-authored various publications. Pet. Ex. 29 at 3-4.

b. Opinion

Dr. Aradillas opined that “to a reasonable degree of medical certainty,” petitioner developed a peripheral neuropathy, specifically CIAP, following his November 2013 immunizations. Pet. Ex. 28 at 2, 8. He concluded that “it [was] more likely than not that [petitioner’s] vaccination[s] in November 2013 were a substantial factor in the onset of his peripheral neuropathy in late December” 2013. Id. at 16.

i. Althen Prong One

Dr. Aradillas opined that the immune mediated mechanism at play is molecular mimicry. He explained that CIAP is a diagnosis with “trustworthy support” for autoimmune mechanisms, including molecular mimicry.²² Pet. Ex. 50 at 4. The same mechanism is seen in a demyelinating neuropathy, like GBS and chronic inflammatory demyelinating polyneuropathy (“CIDP”), where immune activation causes antibodies to attack the peripheral nerves. Pet. Ex. 28 at 4. Like Dr. Grewal, Dr. Aradillas opined that molecular mimicry is one example of a mechanism that explains how the “antibodies are generated after post-infectious exposure or exposure with sterile antigens.” Id. at 4.

Autoimmune neuropathies, as Dr. Aradillas explained, “present with a broad range of symptoms.” Pet. Ex. 28 at 4. Relying on Chroni et al.,²³ Dr. Aradillas noted “[m]ost cases of pure sensory neuropathy are considered idiopathic and categorized as chronic axonal neuropathy or cryptogenic polyneuropathy.” Id. (citing Pet. Ex. 33 at 1). When demyelinating features are

²¹ Petitioner filed two expert reports authored by Dr. Aradillas. Pet. Exs. 28, 50.

²² Dr. Aradillas discusses other potential mechanisms as well. See Pet. Ex. 28. However, for clarity, the undersigned addresses only those mechanisms applicable to her analysis.

²³ Elisabeth Chroni et al., Pure Sensory Chronic Inflammatory Polyneuropathy: Rapid Deterioration After Steroid Treatment, 15 BMC Neurology 1 (2015).

not shown on electrodiagnostic tests, “patients are considered to have an axonal neuropathy.” Id. (quoting Pet. Ex. 35 at 2).²⁴

In a subgroup of CIAP patients, “molecular mimicry is a reliable mechanism to explain axonal destruction of large or small sensory fibers.” Pet. Ex. 50 at 6. Relying on two articles filed by respondent,²⁵ Dr. Aradillas illustrated the mechanism by using the example of *Campylobacter jejuni*²⁶ (“*C. jejuni*”), a bacterial infection that causes GBS. See id. “[C]ertain antecedent infections cause autoantibodies to be generated that target both the bacteria and human peripheral nerve gangliosides, triggering axonal degeneration and demyelination of the peripheral nerves.” Id. (emphasis omitted). He further explained that “several studies have demonstrated that patients infected with *C. jejuni* [are] more likely to develop an axonal subtype than demyelinating subtype of GBS.” Id. In these patients, the autoimmune reaction is “directed against axonal components.” Id.

In Dalakas, an article cited by Dr. Aradillas and respondent’s expert, Dr. Chaudhry, the author stated that “[a]utoimmune [p]eripheral [n]europathies [] develop when immunologic tolerance to key antigenic sites on the myelin, axon, nodes of Ranvier[,] or ganglionic neurons is lost.” Resp. Ex. D8 at 1. Dalakas added that “[c]urrent evidence supports the notion that . . . autoimmunity is mediated by antibodies directed against myelin antigens, along with autoreactive T cells and macrophages that invade myelin sheath, axonal membranes[,] or the nodes of Ranvier.” Id.

Dr. Aradillas further explained how the nodes of Ranvier are vulnerable to immune-mediated injury. See Pet. Ex. 50 at 8. “[W]hen these ‘gaps’ between the myelin sheath of nerves are disrupted by antiganglioside antibodies without causing demyelination, this is known as nodopathy, as is the case in patients with AMAN, the axonal/motor GBS variant.” Id. (citing Pet. Ex. 36 at 4).²⁷ He opined that this process applies to axonal degeneration in sensory fibers. Id. Dr. Aradillas concluded that “the mechanism of molecular mimicry can cause autoantibodies that . . . cause axonal degeneration by direct binding to components of axonal membrane or targeting nodal[] structures to cause axonal degeneration and/or dysfunction,” representing a “sound and reliable theory that is [] well recognized.” Id. at 9.

²⁴ Russell L. Chin et al., Sensory CIDP Presenting as Cryptogenic Sensory Polyneuropathy, 9 J. Peripheral Nervous Sys. 132 (2004).

²⁵ Kishan Kumar Nyati & Roopanshi Nyati, Role of *Campylobacter Jejuni* Infection in the Pathogenesis of Guillain-Barré Syndrome: An Update, 2013 BioMed Rsch. Int’l 1 (filed as Resp. Ex. D7); Marinos C. Dalakas, Pathogenesis of Immune-Mediated Neuropathies, 1852 Biochimica et Biophysica Acta 658 (2015) (filed as Resp. Ex. D8).

²⁶ *Campylobacter jejuni* is “a species that is a common cause of enteric campylobacteriosis in humans.” Campylobacter Jejuni, Dorland’s Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=62516> (last visited on Feb. 8, 2021).

²⁷ Antonino Uncini & Satoshi Kuwabara, Nodopathies of the Peripheral Nerve: An Emerging Concept, 86 J. Neurology Neurosurgery & Psychiatry 1186 (2015).

In further support of his opinions, Dr. Aradillas discussed autoimmune T cells and explained that “T and B cell receptors are generated in response to activation” and can “generat[e] self-reactive (auto)antibodies and autoimmune T cells.” Pet. Ex. 28 at 13-15. Patients with idiopathic sensory neuropathies have been found to have dominant T-cell clones and various antibodies associated with immune-mediated peripheral neuropathies. Id. at 14 (citing Pet. Ex. 22 at 5).

Dr. Aradillas also cited improvement with immunotherapeutic treatment in support of his opinions. See Pet. Ex. 28 at 5. In Chin et al., the authors reviewed records of eight patients with CIDP who presented with sensory neuropathy and no electrodiagnostic features of demyelination. Pet. Ex. 35 at 1-2. The clinical presentation was consistent with sensory CIDP and the patients “exhibited only minimal abnormalities or changes characteristic of axonal degeneration.” Id. at 3. The authors wrote that there were “reports of patients with predominantly large fiber sensory neuropathy, or chronic idiopathic ataxic neuropathy, without electrophysiologic evidence of demyelination, that improved following IVIg therapy.” Id. at 5. Dr. Aradillas opined this was “circumstantial evidence that the neuropathy was immune mediated.” Pet. Ex. 28 at 5.

Moreover, Dr. Aradillas noted that CIAP has been recognized in medical literature as an atypical variant form of CIDP. Pet. Ex. 28 at 5 (citing Pet. Ex. 51 at 2).²⁸ He explained that CIAP describes “patients with a slowly progressive type of peripheral neuropathy, like CIDP which is also a slowly progressive peripheral neuropathy that can be chronically progressive or relapsing with stepwise progression.” Pet. Ex. 50 at 7. Additionally, “[b]oth CIAP and CIDP can involve sensory-only fibers, not exclusive of sensory fiber size, as both large and small sensory fibers are implicated in clinical symptoms.” Id.

In his supplemental report, Dr. Aradillas clarified that he does not suggest that petitioner suffered from GBS or CIDP, but his opinion was influenced by the immune-mediated mechanisms seen in GBS and CIDP cases. Pet. Ex. 50 at 4.

Another causal mechanism proposed by Dr. Aradillas involves aberrant inflammatory signaling that leads to axonal degeneration. Pet. Ex. 28 at 8. The inflammatory response begins with a pro-inflammatory phase, where cytokines, chemokines, and other mediators are secreted, which “can increase nerve excitability, damage myelin, and alter the blood-nerve barrier.” Id. at 9. Vaccines elicit an immune response by “provid[ing] sufficient danger signals through vaccine antigens and/or adjuvants to trigger an inflammatory response.” Id. at 10. Cells already present in the tissues serve as the first line of protection against a foreign pathogen, such as infection, vaccination, or injury. Id. at 9. Once these cells are activated, they are “recruited to the involved area and can invade the nerve through a disrupted blood nerve barrier.” Id.

²⁸ David Schafflick et al., Novel Pathomechanisms in Inflammatory Neuropathies, 14 J. Neuroinflammation 1 (2017).

Citing Obermoser et al.,²⁹ Dr. Aradillas concluded that the administration of the flu or pneumococcal vaccine can cause a local inflammatory reaction mediated by innate immune system cells. Pet. Ex. 28 at 10. Obermoser et al. elicited gene expression profiles within hours of the flu or Pneumovax23³⁰ vaccination and found “vaccines act as potent activators of the innate immune system and that responses can be detected in the blood in vivo within hours following administration.” Pet. Ex. 43 at 2, 5.

ii. Althen Prong Two

Dr. Aradillas agreed that petitioner’s clinical picture and testing was consistent with the diagnosis of CIAP.³¹ Pet. Ex. 28 at 8.

Further, he opined that there is a “logical sequence of events that align with reliable studies from the literature implicating [petitioner’s] [flu] and Prevnar vaccines [as] most likely causal.” Pet. Ex. 50 at 17. First, he stated that onset was appropriate, described in more detail below. Id.; Pet. Ex. 28 at 15-16.

Second, Dr. Aradillas opined that petitioner’s “immune system was in the process of forming an adaptive immune response to his [flu] vaccine when he received his Prevnar 13 vaccine 10 days later.” Pet. Ex. 50 at 17. He stated that the Prevnar vaccine “would have induced additional innate inflammatory responses within [one] day.” Pet. Ex. 28 at 15. Five days after his Prevnar vaccination, petitioner complained of burning pain in his throat that Dr. Aradillas believed could have been induced by the aberrant immune stimulation occurring due to vaccination. Id.; Pet. Ex. 50 at 17. He opined that “[t]his timing would overlap when potential molecular mimics could be the result of either vaccine and produce additional inflammatory stimulus.” Pet. Ex. 50 at 17. He found that the combination of burning pain in petitioner’s throat³² with suspicion of infectious causes and the resolution of petitioner’s symptoms with a Medrol dose pack indicated ongoing inflammation in petitioner when his body was working to develop antibodies to the flu and Prevnar vaccines. Pet. Ex. 28 at 15. Therefore, “[i]t is

²⁹ Gerlinde Obermoser et al., Systems Scale Interactive Exploration Reveals Quantitative and Qualitative Differences in Response to Influenza and Pneumococcal Vaccines, 38 Immunity 831 (2013).

³⁰ This is a different pneumococcal vaccination than the one administered to petitioner.

³¹ In his reports, Dr. Aradillas also discussed small fiber neuropathy and he opined that it remains a differential diagnosis. Pet. Ex. 50 at 10. Regardless, this does not appear to impact his opinions as to causal mechanisms or to causation.

³² In his supplemental report, he clarified that he does not opine that petitioner’s sore throat was caused by his vaccinations, but was due to his new mouth guard. Pet. Ex. 50 at 15. Petitioner’s throat symptoms resolved after he took the prednisone; however, “by this time the axonal damage to [petitioner’s] peripheral sensory fibers had already been initiated” and his new complaints of numbness and tingling were indicative of the development of a peripheral neuropathy. Id. at 16-17.

biologically plausible that an aberrant inflammatory reaction could have resulted in an adverse production of autoimmune antibodies, autoimmune T cells, or directly induced small fiber damage in [petitioner].” Id. at 15-16.

iii. Althen Prong Three

With regard with Althen Prong Three, Dr. Aradillas found that there is a “biologically plausible proximate temporal relationship” between the onset of petitioner’s symptoms and his vaccinations. Pet. Ex. 28 at 16. He opined that petitioner’s bilateral numbness and tingling began around December 28, 2013, or around 45 days after the administration of his Prevnar 13 vaccination, which is within the six week time frame that “T lymphocytes have been described [as] being present after nerve injury in peripheral nerves.”³³ Id. at 16. This time frame is also within the six to eight weeks GBS was first observed after the swine flu vaccine. Pet. Ex. 50 at 17.

Given the fact that petitioner purchased the Prilosec on December 26, 2013, and that he began taking it on December 27, 2013, does not change Dr. Aradillas’ opinion as to onset or vaccine causation. Pet. Ex. 50 at 17. “Changing that timeframe by a day or so” does not make a difference, as onset is still within the six to eight weeks recognized as acceptable by the literature. Id.

Dr. Aradillas concluded it is more likely than not that petitioner’s November 2013 vaccinations were a substantial factor in the onset of his peripheral neuropathy in December 2013. Pet. Ex. 28 at 16.

3. Respondent – Dr. Vinay Chaudhry³⁴

a. Background and Qualifications

Dr. Chaudhry is board certified in neurology, neuromuscular diseases, electrodiagnostic medicine, and clinic neurophysiology. Resp. Ex. A at 1. Dr. Chaudhry obtained his M.B. and B.S. in 1980 in India. Resp. Ex. B at 2. He then completed an internship and various residencies and fellowships from 1980 to 1989. Id. at 2-3. Dr. Chaudhry is currently a Professor of Neurology at John Hopkins University School of Medicine and the Co-Director of the EMG Laboratory at John Hopkins Hospital. Resp. Ex. A at 1. He has an active clinical practice and sees around 2000 patients per year. Id. Dr. Chaudhry has authored or co-authored over 200 publications. Resp. Ex. B at 3-20.

b. Opinion

Dr. Chaudhry agreed with the diagnosis of mild, chronic, sensory, small and large fiber neuropathy, and because most other known etiologies were excluded, he agreed that petitioner’s

³³ It is not clear what study Dr. Aradillas is referencing here.

³⁴ Respondent filed three expert reports authored by Dr. Chaudhry. Resp. Exs. A, C-D.

condition appears consistent with typical CIAP. Resp. Ex. A at 6. However, he opined that petitioner's flu and Prevnar vaccinations played no role in causing petitioner's polyneuropathy. Id.

i. Althen Prong One

While Dr. Chaudhry did not take issue with the mechanism of molecular mimicry, *per se*, he disagreed with petitioner's experts' use of that and other mechanisms described as causal in GBS and CIDP, because petitioner was not diagnosed with either of those conditions. He distinguished CIAP from those conditions. Dr. Chaudhry explained that GBS is monophasic with symptoms peaking at less than four weeks followed by improvement. Resp. Ex. A at 7. GBS typically presents with ascending paralysis and areflexia, that begins in the proximal and distal muscles and progresses to the facial, bulbar, and diaphragm muscles. Id.; Resp. Ex. C at 3. Tests show demyelinating features and abnormal motor results. Resp. Ex. A at 7. With CIDP, there is no ascending weakness or involvement of the diaphragm, bulbar, or other cranial nerves. Resp. Ex. C at 3.

Dr. Chaudhry agreed with Dr. Aradillas that "most cases of pure sensory neuropathy are considered idiopathic and categorized as chronic axonal neuropathy or cryptogenic polyneuropathy." Resp. Ex. D at 4. However, he argued Dr. Aradillas provided no evidence to support his suggestion that CIAP is a variant of CIDP. Id.

Dr. Chaudhry addressed Dr. Aradillas' proposed mechanisms as they related to the facts and circumstance of this case. See Resp. Ex. D at 5-6. First, he noted that Dr. Aradillas' discussion regarding the mechanism found in neuropathic pain is not relevant because petitioner did not have neuropathic pain, or suffer from any disease associated with neuropathic pain. Id. at 5. Regarding Dr. Aradillas' opinion that antibodies may have caused an inflammatory response or a non-inflammatory response, Dr. Chaudhry opined that petitioner did not have antibodies, nor was there evidence of channelopathy or an inflammatory neuropathy. Id. Additionally, "[n]o autoantibodies have been conclusively shown to be induced by vaccination and to have induced neuropathy." Id.

Dr. Chaudhry next argued that Dr. Aradillas "equate[d] all immune neuropathies and lump[ed] them together" when immune mechanisms are highly specific. Resp. Ex. D. at 5-6. Within GBS, specifically, "the pathogenesis, the antibodies involved, the preceding infection, [and] the site of damage . . . differ depending on the phenotype." Id. at 6. Similarly, CIDP encompasses several different chronic demyelinating neuropathies, each with different presentations, pathogenesis, treatments, and prognoses. Id.

ii. Althen Prong Two

In his second expert report, Dr. Chaudhry discussed the types of peripheral neuropathies and explained that "the clinical presentation, the time course of onset, the clinical progression, individual fiber-types affected, the distribution of involvement, the electrodiagnostic findings, the etiology, and the laboratory findings" are instructive in determining the etiology of petitioner's peripheral neuropathy. Resp. Ex. C at 1-2. Dr. Chaudhry reviewed each factor as it

related to petitioner's case and found petitioner had (1) a chronic time course; (2) a mildly progressive clinical course; (3) "only sensory fiber involvement with tingling and numbness with both small and large fiber involvement but no motor or autonomic involvement;" (4) distribution that was "length dependent" and confined to the feet and calves; (5) electrodiagnostic findings consistent with "a very mild large fiber sensory axonal neuropathy," which "is not seen with any immune neuropathies;" and (6) normal blood tests with no "laboratory tests to suggest an autoimmune etiology." Id. at 2-4 (emphasis omitted). Based on these factors and findings, Dr. Chaudhry concluded petitioner "had mild sensory length dependent axonal neuropathy of long standing nature, a pattern seen classically with cryptogenic idiopathic neuropathies." Id. at 5.

Dr. Chaudhry disagreed with petitioner's experts that the "vaccinations were a substantial cause or trigger" in his polyneuropathy. Resp. Ex. A at 7 (internal quotation marks omitted). Dr. Chaudhry opined that petitioner's vaccinations had no causative role in the development of his polyneuropathy for two reasons: (1) the temporal relationship between vaccinations and onset was over seven weeks and not medically appropriate; and (2) there is no evidence petitioner developed an acute immune neuropathy, such as GBS, because his symptoms fluctuated and progressed over three years, his reflexes were preserved, he had no weakness, and no features of GBS were shown in his nerve conduction study. Id. at 6.

Regarding Dr. Grewal's discussion of GBS in this case, Dr. Chaudhry opined petitioner had no features consistent with GBS. Resp. Ex. A at 7. Petitioner's symptoms were progressive, not monophasic, for over three years. Id. Petitioner did not have weakness, had normal strength, and his reflexes were brisk, not absent. Id. Additionally, petitioner's EMG showed axonal and sensory findings, and no demyelinating features. Id. Lastly, Dr. Chaudhry noted that none of petitioner's treating physicians considered conducting a spinal tap (to test cerebrospinal fluid) or treating petitioner with immune medications, which would be typical in a GBS case. Id.

In his supplemental report, Dr. Chaudhry examined petitioner's distribution of symptoms and concluded that petitioner did not have any features found in immune neuropathies, such as GBS or CIDP. Resp. Ex. C at 3. Although both of petitioner's experts suggested that petitioner may have had autoantibodies for several weeks after vaccination,³⁵ Dr. Chaudhry argued there was no evidence that petitioner had any antibodies after vaccination or that his condition was autoimmune.³⁶ Resp. Ex. A at 8; Resp. Ex. D at 5. He further argued that even if there were antibodies present, there was no evidence these antibodies could continue to cause damage for the next three years. Resp. Ex. A at 8.

³⁵ Specifically, Dr. Grewal stated "[i]t is most likely that [petitioner] responded to his vaccinations by producing antibodies that then attacked his axonal nerves," and thus, "axonal damage was occurring from the production of autoantibodies for several weeks before [petitioner] began to feel numbness and tingling." Pet. Ex. 19 at 7. Dr. Aradillas suggested "an aberrant inflammatory reaction could have resulted in an adverse production of autoimmune antibodies, autoimmune T cells, or direct induced small fiber damage in [petitioner]." Pet. Ex. 28 at 15-16.

³⁶ The onset of petitioner's polyneuropathy was approximately December 29, 2013, and antibodies were not tested for until June 2014.

In his second supplemental report, Dr. Chaudhry addressed Dr. Aradillas' suggestion that petitioner suffered from an atypical sensory variant of GBS or CIDP and reiterated that petitioner showed no features of GBS or CIDP. Resp. Ex. D. at 3-4. Dr. Chaudhry explained that a patient has sensory GBS when they have "clinically pure acute sensory neuropathy [] with electrophysiological evidence of demyelination in motor and sensory fibers, or more rarely only in sensory fibers." Id. at 3. He then went through each feature of sensory GBS and noted petitioner did not show, or was not tested for, each feature. See id. at 3-4.

CIDP is a "sensorimotor demyelinating polyneuropathy that is characterized clinically by progressive weakness and areflexia; electrophysiologically by features of demyelination; laboratory features of albuminocytological dissociation in cerebrospinal fluid; and pathology of inflammation and demyelination/remyelination on nerve biopsy." Resp. Ex. D at 4. Similar to his analysis as to GBS, Dr. Chaudhry noted petitioner had none of the clinical, electrophysiological, or laboratory features of CIDP, and petitioner did not show, or was not tested for, the features of CIDP. Id. Additionally, Dr. Chaudhry discussed the diagnostic criteria of a small fiber neuropathy and found petitioner did not meet the criteria. Id. at 2-3.

Overall, Dr. Chaudhry concluded that petitioner developed a mild, chronic, sensory, small and large fiber neuropathy, the etiology of which is idiopathic and not due to vaccination. Resp. Ex. D at 10. Even though he opined that the cause of petitioner's neuropathy is idiopathic, and that testing did not reveal evidence of diabetes, glucose intolerance, or B12 deficiency, he maintained that the possibility of glucose intolerance and B12 deficiency cannot be excluded. Id.; Resp. Ex. A at 6. Although blood tests were normal, he argued that a "B12 deficiency [could not] be ruled out since [petitioner] was on replacement B12 medications and [had a] skin rash and arthropathy [that] suggest[ed] that more extensive connective tissue disease." Resp. Ex. C at 4 (emphasis omitted).

iii. Althen Prong Three

Regarding Althen Prong Three, Dr. Chaudhry opined the timeframe between vaccinations and onset is not medically appropriate. Resp. Ex. A at 6. Unlike Dr. Grewal and Dr. Aradillas, Dr. Chaudhry found petitioner's neurological symptoms began mid-January 2014, not in December 2013. Resp. Ex. A at 5, 7. For support, Dr. Chaudhry cited petitioner's January 16, 2014 visit, where he first described numbness and tingling. Id. at 7 (citing Pet. Ex. 11 at 3). Additionally, he noted that Dr. Rao documented onset of symptoms in January 2014.³⁷ Id. (citing Pet. Ex. 17 at 6).

³⁷ Dr. Rao documented that "[petitioner] began experiencing tingling and numbness of his toes involving the ventral surfaces bilaterally since January 2014. He was prescribed [medication] for GERD in January 2014, [and] he thinks his symptoms began a few days after." Pet. Ex. 8 at 18. Pharmacy records and an affidavit filed by petitioner show that petitioner began taking the relevant medication on December 27, 2013. Pet. Ex. 24; Pet. Ex. 27 at ¶¶ 2-3.

V. DISCUSSION

A. Standards for Adjudication

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 10(a). “Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award ‘vaccine-injured persons quickly, easily, and with certainty and generosity.’” Rooks v. Sec’y of Health & Hum. Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner’s burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec’y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. Bunting v. Sec’y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, petitioner must prove that the vaccine was “not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury.” Moberly, 592 F.3d at 1321 (quoting Shyface v. Sec’y of Health & Hum. Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); see also Pafford v. Sec’y of Health & Hum. Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner who satisfies this burden is entitled to compensation unless respondent can prove, by a preponderance of the evidence, that the vaccinee’s injury is “due to factors unrelated to the administration of the vaccine.” § 13(a)(1)(B).

B. Factual Issues

A petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding his claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. See Burns v. Sec’y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a special master must decide what weight to give evidence including oral testimony and contemporaneous medical records). Contemporaneous medical records are presumed to be accurate. See Cucuras v. Sec’y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). To overcome the presumptive accuracy of medical records, a petitioner may present testimony which is “consistent, clear, cogent, and compelling.” Sanchez v. Sec’y of Health & Hum. Servs., No. 11-685V, 2013 WL 1880825, at *3 (Fed. Cl. Spec. Mstr. Apr. 10, 2013) (citing Blutstein v. Sec’y of Health & Hum. Servs., No. 90-2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)).

There are situations in which compelling testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. Campbell v. Sec’y of Health & Hum. Servs., 69 Fed. Cl. 775, 779 (2006) (“[L]ike any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking.”); Lowrie v. Sec’y of Health & Hum. Servs., No. 03-1585V, 2005 WL 6117475, at *19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005) (“[W]ritten records which are, themselves, inconsistent, should be accorded less deference than

those which are internally consistent.” (quoting Murphy v. Sec’y of Health & Hum. Servs., 23 Cl. Ct. 726, 733 (1991), aff’d per curiam, 968 F.2d 1226 (Fed. Cir. 1992))). Ultimately, a determination regarding a witness’s credibility is needed when determining the weight that such testimony should be afforded. Andreu v. Sec’y of Health & Hum. Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); Bradley v. Sec’y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

Despite the weight afforded medical records, special masters are not bound rigidly by those records in determining onset of a petitioner’s symptoms. Valenzuela v. Sec’y of Health & Hum. Servs., No. 90-1002V, 1991 WL 182241, at *3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); see also Eng v. Sec’y of Health & Hum. Servs., No. 90-1754V, 1994 WL 67704, at *3 (Fed. Cl. Spec. Mstr. Feb. 18, 1994) (Section 13(b)(2) “must be construed so as to give effect also to § 13(b)(1) which directs the special master or court to consider the medical records (reports, diagnosis, conclusions, medical judgment, test reports, etc.), but does not require the special master or court to be bound by them”).

C. Causation

To receive compensation through the Program, petitioner must prove either (1) that petitioner suffered a “Table Injury”—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that he received, or (2) that petitioner suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano v. Sec’y of Health & Hum. Servs., 440 F.3d 1317, 1319-20 (Fed. Cir. 2006). Because petitioner does not allege that he suffered a Table Injury, he must prove that a vaccine he received caused his injury. To do so, he must establish, by preponderant evidence: (1) a medical theory causally connecting the vaccine and his injury (“Althen Prong One”); (2) a logical sequence of cause and effect showing that the vaccine was the reason for his injury (“Althen Prong Two”); and (3) a showing of a proximate temporal relationship between the vaccine and his injury (“Althen Prong Three”). § 13(a)(1); Althen, 418 F.3d at 1278.

The causation theory must relate to the injury alleged. The petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be “legally probable, not medically or scientifically certain.” Knudsen v. Sec’y of Health & Hum. Servs., 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioner cannot establish entitlement to compensation based solely on his assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether petitioner is entitled to compensation, the special master shall consider all material in the record, including “any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation.” § 13(b)(1)(A). The undersigned must weigh the submitted evidence and the testimony of the parties’ proffered experts and rule in petitioner’s favor when the evidence weighs in his favor. See Moberly, 592 F.3d at 1325-26 (“Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence.”); Althen, 418 F.3d at 1280 (noting that “close calls” are resolved in petitioner’s favor).

“Expert medical testimony which merely expresses the possibility—not the probability—of the occurrence of a compensable injury is insufficient, by itself, to substantiate the claim that such an injury occurred.” LaCour v. Sec’y of Health & Hum. Servs., No. 90-316V, 1991 WL 66579, at *5 (Fed. Cl. Spec. Mstr. Apr. 15, 1991); accord Burns v. Sec’y of Health & Hum. Servs., No. 90-953V, 1992 WL 365410, at *6 (Fed. Cl. Spec. Mstr. Nov. 6, 1992), aff’d, 3 F.3d 415 (Fed. Cir. 1993). The Federal Circuit has likewise made clear that the mere possibility of a link between a vaccination and a petitioner’s injury is not sufficient to satisfy the preponderance standard. Moberly, 592 F.3d at 1322 (emphasizing that “proof of a ‘plausible’ or ‘possible’ causal link between the vaccine and the injury” does not equate to proof of causation by a preponderance of the evidence); Waterman v. Sec’y of Health & Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying petitioner’s motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard). While certainty is by no means required, a possible mechanism does not rise to the level of preponderance. Id.; see also de Bazan v. Sec’y of Health & Hum. Servs., 539 F.3d 1347, 1351 (Fed. Cir. 2008).

D. Causation Analysis

1. Althen Prong One

Under Althen Prong One, petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1375; Pafford, 451 F.3d at 1355-56. Petitioner’s theory of causation need not be medically or scientifically certain, but it must be informed by a “sound and reliable” medical or scientific explanation. Boatmon v. Sec’y of Health & Hum. Servs., 941 F.3d 1351, 1359 (Fed. Cir. 2019); see also Knudsen, 35 F.3d at 548; Veryzer v. Sec’y of Health & Hum. Servs., 98 Fed. Cl. 214, 223 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both “relevant” and “reliable”). If petitioner relies upon a medical opinion to support his theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen v. Sec’y of Health & Hum. Servs., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (“The special master’s decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories.”); Perreira v. Sec’y of Health & Hum. Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an “expert opinion is no better than the soundness of the reasons supporting it” (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

The undersigned finds that petitioner has provided preponderant evidence of Althen Prong One, that the flu vaccine can cause CIAP, based on the petitioner’s expert reports and opinions and supportive medical literature.³⁸

Both Dr. Grewal and Dr. Aradillas proffer molecular mimicry as a likely causal mechanism of CIAP. The most straightforward analysis was offered by Dr. Grewal, who explained that this form of neuropathy is immune-mediated, and believed to be triggered by

³⁸ Although Dr. Aradillas made an interesting point about the body’s response to Prevnar vaccine after a flu vaccine, this point was not well-developed, and the undersigned does not find that Prevnar played a causal role based on the evidence in this case.

cross-reactivity of immune cells in the vaccine, resulting in an “inadvertent immune response against self cells.” Pet. Ex. 19 at 5.

In support of the proffered mechanism, Dr. Grewal cites at least two articles that discuss the etiology of CIAP. The first of these is authored by Hughes et al., where the authors state that one of the causes is thought to be “autoimmune responses to axonal antigens.” Pet. Ex. 21 at 2. In Zis et al., the authors observe that “[a]n underlying immunological process has been proposed after circulating T cell clones were found in [CIAP] patients.” Pet. Ex. 22 at 5. They explain that studies have shown that some patients with CIAP respond to immunosuppressive therapy. Id. While acknowledging that more studies are needed to fully understand the etiology, they conclude that CIAP may be an immune-mediated neuropathy. Id.

In Klein,³⁹ an article cited by Dr. Aradillas, the authors discuss the architecture of the nerve. See Pet. Ex. 40. Klein states, “[a]ccumulating evidence suggests that immune-mediated events may start as a result of molecular mimicry targeting different specific architectures within [the] nerve.” Id. at 3. The “blood-nerve interface” is “dynamic and variably permeable to immune-mediating cells.” Id. The “blood-nerve interface” allows T cells and B lymphocytes to cross, allowing for direct contact with nerve tissues. Id. Additionally, “the nodes of Ranvier, where Schwann cell microvilli have loose connections to the axon, there is additional immune susceptibility.” Id.

Literature filed by respondent also supports the proffered immune-mediated theory. In Dalakas, the author states,

Autoimmune Peripheral Neuropathies (APN) develop when immunologic tolerance to key antigenic sites on the myelin, axon, nodes of Ranvier[,] or ganglionic neurons is lost. Current evidence supports the notion that in APN the autoimmunity is mediated by antibodies directed against myelin antigens, . . . axonal membranes[,] or the nodes of Ranvier.

Resp. Ex. D8 at 1.

The above articles corroborate the opinions of Dr. Grewal and Dr. Aradillas, and establish that molecular mimicry has been proposed as a mechanism of immune-mediated injury in peripheral neuropathies characterized by axonal injury.

The mechanism of molecular mimicry has been accepted numerous times in the Vaccine Program as a sound and reliable medical theory, particularly with regard to peripheral

³⁹ Christopher J. Klein, Autoimmune-Mediated Peripheral Neuropathies and Autoimmune Pain, 133 Handbook Clinical Neurology 417 (2016).

neuropathies.⁴⁰ Based on the expert opinions here, supported by medical literature, molecular mimicry is thought to be the causal mechanism be at play when polyneuropathy is caused by axonal injury after the flu vaccine.

Dr. Chaudhry takes issue with the comparisons drawn by Dr. Aradillas between small fiber neuropathy, GBS, and CIDP, and explains why petitioner did not have these other conditions. Dr. Aradillas, however, uses these other forms of neuropathy to illustrate the characteristics of immune-mediated illnesses, not to suggest that petitioner had these diagnoses.

In contrast, Dr. Chaudhry categorizes peripheral neuropathy cases into ten types, and he asserts that pathogenesis can be determined based on a set of factors. These include the time course, clinical course, type of fiber affected, distribution of symptoms, EMG findings, laboratory and diagnostic tests, and response to different types of treatment. As to each category, he concludes that petitioner's findings do not fit an immune etiology. This approach does not seem well-grounded in the medical literature that the parties cited. Further, it is erroneous to conclude that because a peripheral neuropathy is due to axonal injury, it cannot be immune-mediated, especially where an immune origin has been suggested in the medical literature.

Accordingly, the undersigned finds that petitioner has set forth a sound and reliable medical theory, satisfying Althen Prong One.

2. Althen Prong Two

Under Althen Prong Two, petitioner must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278). "Petitioner must show that the vaccine was the 'but for' cause of the harm . . . or in other words, that the vaccine was the 'reason for the injury.'" Pafford, 451 F.3d at 1356 (internal citations omitted).

In evaluating whether this prong is satisfied, the opinions and views of the vaccinee's treating physicians are entitled to some weight. Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 ("[M]edical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" (quoting Althen, 418 F.3d at 1280)). Medical records are generally viewed as trustworthy evidence since they are created contemporaneously with the treatment of the vaccinee. Cucuras, 993 F.2d at 1528. The

⁴⁰ See, e.g., Drobbin v. Sec'y of Health & Hum. Servs., No. 14-225V, 2020 WL 3799206, at *16-17 (Fed. Cl. Spec. Mstr. Jan. 21, 2020) (finding petitioner established by preponderant evidence that the flu vaccine can cause the development of some peripheral neuropathies, including the type of small fiber neuropathy petitioner suffers from, via molecular mimicry); Auch v. Sec'y of Health & Hum. Servs., No. 12-673V, 2017 WL 1034396, at *12 n.19, *20 (Fed. Cl. Spec. Mstr. Jan. 13, 2017) (finding petitioner provided preponderant evidence that the flu vaccine can cause a neuropathy through molecular mimicry, satisfying Althen prong one); Doe/06 v. Sec'y of Health & Hum. Servs., No. [redacted]V, 2007 WL 3120297 (Fed. Cl. Spec. Mstr. Oct. 18, 2017) (granting entitlement in a flu/small fiber neuropathy case).

petitioner need not make a specific type of evidentiary showing, i.e., “epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect.” Capizzano, 440 F.3d at 1325. Instead, petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

The parties have agreed that petitioner’s diagnosis is CIAP. Further, the undersigned has found that petitioner has proven that the flu vaccine can cause CIAP. Thus, the next issue is whether petitioner’s flu vaccination caused his CIAP, given the facts and circumstances present here. The undersigned finds that there is preponderant evidence to establish a “logical sequence of cause and effect showing that the vaccination was the reason for the injury.” Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278).

There are three reasons for the undersigned’s finding as to Althen Prong Two. First, petitioner’s treating neurologist opined that the cause of petitioner’s CIAP could be immunological (on his differential list), and his PCP suggested that his neuropathy might possibly be related to his flu vaccine. Second, a thorough workup found no alternative cause for petitioner’s condition. Lastly, there is a temporal association, as discussed in the Althen Prong Three analysis.

Petitioner’s treating neurologist, Dr. Rao, conducted a thorough workup of petitioner in June 2014, performing an MRI and diagnostic laboratory tests. After reviewing the diagnostic testing, he opined that there were three differential causes: metabolic, toxic, or immunological. Thus, of the potential causes, an immune-mediated cause was specifically enumerated by Dr. Rao.

Dr. Adler, petitioner’s PCP, also considered an immune-mediated cause, specifically the possibility of an association with petitioner’s vaccinations. While an opinion stated as a possibility, by itself, does not constitute preponderant evidence, the fact that Dr. Adler, a treating physician, considered an immune-mediated cause presents some evidence in support of vaccine causation. See Moberly, 592 F.3d at 1322; Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326.

With regard to alternative causes for petitioner’s condition, including metabolic causes, Dr. Chaudhry explained that diabetes or glucose intolerance were not likely causes of petitioner’s polyneuropathy, although he did not exclude them, because testing revealed that petitioner was not diabetic, and he did not have glucose intolerance. As for medication, or toxins, Dr. Chaudhry considered whether petitioner’s neuropathy was a side effect of his omeprazole. Dr. Chaudhry explained that PPI drugs, like omeprazole, can cause Vitamin B12 deficiencies, which can contribute to peripheral neuropathy. However, petitioner had a normal B12, and so Dr. Chaudhry concluded that this cause was unlikely, but did not exclude it. Neither opinion reaches the level of more likely than not.

Dr. Chaudhry cites Visser et al.⁴¹ to argue petitioner's polyneuropathy is more common in those who are 75-79 years old. Resp. Ex. C at 5-6. Visser et al. found the incidence rates of polyneuropathies increase with age and specifically found "a prominent increase in the proportion of [CIAP] with advancing age." Resp. Ex. C2 at 3. Although petitioner was seventy years old at the time of vaccination, none of petitioner's treating physicians attributed his polyneuropathy to aging, and thus, the undersigned does not find this argument persuasive.

Testing "excluded most other known etiologies."⁴² Resp. Ex. A at 6. Therefore, Dr. Chaudhry concluded that petitioner's neuropathy was idiopathic, stating that "after exhaustive investigation no clear cause is found in about 25% of patients." *Id.* (citing Pet. Ex. 21 at 1). Dr. Grewal agreed with Dr. Chaudhry that no cause was identified based on petitioner's testing. However, he opined that petitioner's vaccinations were the likely cause of his illness because (1) no other trigger, including no infectious trigger, of petitioner's symptoms was found and (2) immune-mediated nerve damage is a known cause of neuropathies.

Dr. Chaudhry also suggested that because petitioner did not have antibodies, his condition was not immune-mediated. On this point, Dr. Chaudhry referenced Zis et al., which describes three patients who had antibodies, "one for sulfated glucuronyl paragloboside (SGPG), one for asialo-GM1, and one for anti-Hu." Resp. Ex. A at 8 (citing Pet. Ex. 22 at 5). However, petitioner's medical records only show that he was tested for anti-Hu antibodies, and not SGPG or asialo-GM1 antibodies. Neither Zis et al. nor Dr. Chaudhry explained the significance of these antibodies. Further, the authors of the article did not reach any conclusion about the paucity of antibodies in the majority of patients (54 out of 57 patients did not have antibodies) in the study who had CIAP; instead, after reviewing the relevant literature, they conclude "[i]t is plausible that a number of patients with CIAP may have an immune mediated neuropathy." Pet. Ex. 22 at 5.

Further, although petitioner's test results for IgG, IgA,⁴³ and IgM were not elevated, these tests were done more than six months after onset of his condition. Because testing was not done earlier, it is not possible to know whether petitioner had abnormal antibodies. IgM antibodies, for example, "are produced as a body's first response to a new infection," and "increase for several weeks and then decline as IgG production begins." Immunoglobulins (IgA, IgG, IgM),

⁴¹ Nora A. Visser et al., Incidence of Polyneuropathy in Utrecht, the Netherlands, 84 *Neurology* 259 (2015).

⁴² Dr. Chaudhry opined that petitioner's "[r]ash and arthropathy raises the possibility of a connective disease associated vasculopathy." Resp. Ex. A at 6. However, opinions based on possibilities are insufficient to establish causation. See, e.g., Moberly, 592 F.3d at 1322; de Bazan, 539 F.3d at 1351; Burns, 1992 WL 365410, at *6; LaCour, 1991 WL 66579, at *5. Further, petitioner's rash in December 2013 was determined to be caused by androgel. Pet. Ex. 3 at 8. There is a reference to a rash and arthropathy in petitioner's December 2016 visits to Dr. Gordon. Pet. Ex. 9 at 21-22. None of petitioner's treating physicians raised this concern and they agreed that petitioner developed a polyneuropathy.

⁴³ Petitioner's IgA was low, but none of the experts placed significance on this value.

Merck Manual, <https://www.merckmanuals.com/-/media/Manual/LabTests/Immunoglobulins/IgA/IgG/IgM> (last visited Feb. 10, 2021). And IgG antibodies “are produced during an initial infection or other antigen exposure, rising a few weeks after it begins, then decreasing and stabilizing.” *Id.* Additionally, Nyati and Nyati note “IgA and IgM levels rise in response to infection and remain elevated for 3-4 weeks before declining to baseline levels.” Resp. Ex. D7 at 4. Thus, because testing was done over six months after petitioner’s onset, the results are not reflective of whether the petitioner had antibodies at the time he developed his illness.

For all of these reasons, and taken in the context presented, the presence of or absence of the antibodies here does not appear to be determinative with regard to etiology.

Relying on another article filed by petitioner, Pasnoor et al., which notes that 42% of idiopathic polyneuropathies are hereditary, Dr. Chaudhry suggests that petitioner’s illness could have a genetic cause and notes that genetic testing was not performed on petitioner. Resp. Ex. A at 8-9 (citing Pet. Ex. 23 at 7-8). Raising a genetic cause, however, without more evidence, does not establish an alternative cause of petitioner’s illness nor does it rule out an immune cause.

For all of these reasons, the undersigned finds that petitioner has proven by preponderant evidence that the flu vaccination he received caused his CIAP. Accordingly, petitioner has satisfied Althen Prong Two.

3. Althen Prong Three

Althen Prong Three requires petitioner to establish a “proximate temporal relationship” between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That term has been equated to mean a “medically acceptable temporal relationship.” *Id.* The petitioner must offer “preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disease’s etiology, it is medically acceptable to infer causation-in-fact.” de Bazan, 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also coincide with the theory of how the relevant vaccine can cause the injury alleged (under Althen Prong One). *Id.*; Koehn v. Sec’y of Health & Hum. Servs., 773 F.3d 1239, 1243 (Fed. Cir. 2014); Shapiro v. Sec’y of Health & Hum. Servs., 101 Fed. Cl. 532, 542 (2011), recons. den’d after remand, 105 Fed. Cl. 353 (2012), aff’d mem., 503 F. App’x 952 (Fed. Cir. 2013).

With regard to onset, the pertinent facts are as follows: Petitioner purchased the Medrol pack and omeprazole on December 26, 2013. He began taking the prescribed medication on December 27, 2013. Petitioner returned to Dr. Undavia for a follow-up on January 16, 2014. At that visit, petitioner reported that after the second night of taking omeprazole, he started having numbness and tingling on the bottom of his toes. Petitioner called Dr. Adler on January 17, 2014 and again reported that felt a tingling sensation in his feet after the second day of taking omeprazole.

Based on the medical records and affidavits, the undersigned finds that the onset of petitioner’s neurological condition (numbness and tingling) began two days after petitioner began taking the Medrol pack and omeprazole, which is December 29, 2013.

Dr. Rao's June 5, 2014 record documented that petitioner complained of numbness and tingling in his feet since January 2014, which began "a few days after" he was prescribed medication for GERD. Pet. Ex. 17 at 6. This entry is roughly the same as the undersigned's finding as to onset of December 29, 2013, which is "a few days after" petitioner began taking this medication on December 27. See Pet. Ex. 27 at ¶ 3. The undersigned finds references to onset in January 2014 occurred later in time, approximately six months after petitioner first reported his symptoms. Additionally, the later-in-time records were less specific in nature, referencing January 2014 and "a few days," whereas the first two earlier-in-time records were specific as to onset, stating onset occurred after the second day of taking the medication. Thus, the undersigned finds the most contemporaneous-in-time records to be more reliable regarding onset.

Dr. Grewal opined that the temporal association between petitioner's vaccinations and the onset of his neurological symptoms was medically appropriate. He explained that a medically appropriate timeframe for onset of clinical symptoms after an immune challenge ranges from two to 60 days. Dr. Grewal opined that petitioner reported an onset at the end of December 2013, which is within this timeframe.

Similarly, Dr. Aradillas opined that there was a temporal relationship between petitioner's vaccinations and the onset of his symptoms. He placed onset of petitioner's bilateral numbness and tingling on December 28, 2013, within six to eight weeks after his flu vaccination. He further opined that this time frame was appropriate given the mechanism of molecular mimicry. He also noted that the six-to-eight-week window was within the time frame that GBS was observed after the swine flu vaccine.

However, unlike Dr. Grewal and Dr. Aradillas, Dr. Chaudhry opined in his first report that petitioner's neurological symptoms began mid-January 2014, not end of December 2013, and he found this time frame inappropriate. Specifically, Dr. Chaudhry cited petitioner's January 16, 2014 visit to Dr. Undavia, as the date that petitioner first described numbness and tingling. However, on that visit, petitioner reported that the symptoms occurred after his second dose of omeprazole, and subsequent records showed the date of his second dose to be approximately December 28, 2013. Additionally, Dr. Chaudhry noted that Dr. Rao documented onset of symptoms in January 2014. To be fair, Dr. Chaudhry's opinion as to onset was filed before the petitioner filed his pharmacy records and an affidavit explaining the date that he began taking omeprazole, the medication that formed his frame of reference as to onset of symptoms. However, Dr. Chaudhry did not revise his opinion as to onset after petitioner filed evidence to show when he began taking the medication which petitioner used as a frame of reference to date the onset of the numbness and tingling in his toes.

Although Dr. Chaudhry opines petitioner's onset was in January 2014, the undersigned finds that based on the medical records and affidavit, petitioner's symptoms began on or about December 29, 2013. Using December 29, onset was 55 days after petitioner's flu vaccination. This is an appropriate time frame for an immune-mediated process, particularly the proposed mechanism of molecular mimicry, to occur following vaccination.

Although exceedingly close, 55 days is one day inside the generally accepted timeframe of eight weeks, and five days inside the time frame of 60 days proposed by Dr. Grewal. See Paluck v. Sec’y of Health & Hum. Servs., 786 F.3d 1373, 1383-84 (Fed. Cir. 2015) (finding the “special master [] erred in setting a hard and fast deadline . . . between vaccination and [] onset”).

Thus, the undersigned finds petitioner has provided preponderant evidence of a proximate temporal relationship between petitioner’s flu vaccination and his CIAP.

E. Alternative Causation

Because the undersigned concludes that petitioner has established a prima facie case, petitioner is entitled to compensation unless respondent can put forth preponderant evidence “that [petitioner’s] injury was in fact caused by factors unrelated to the vaccine.” Whitecotton v. Sec’y of Health & Hum. Servs., 17 F.3d 374, 376 (Fed. Cir. 1994), rev’d on other grounds sub nom., Shalala v. Whitecotton, 514 U.S. 268 (1995); see also Walther v. Sec’y of Health & Hum. Servs., 485 F.3d 1146, 1151 (Fed. Cir. 2007). As discussed above in the analysis related to Althen Prong Two, the undersigned finds that respondent failed to establish evidence to show that petitioner’s CIAP was caused by a source other than vaccination. Thus, respondent did not prove by a preponderance of evidence that petitioner’s injury is “due to factors unrelated to the administration of the vaccine.” § 13(a)(1)(B).

VI. CONCLUSION

For the reasons discussed above, the undersigned finds that petitioner has established by preponderant evidence that he is entitled to compensation. A separate damages order will issue.

IT IS SO ORDERED.

s/Nora Beth Dorsey

Nora Beth Dorsey
Special Master